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NEWS	1		Web Page for STN Seminar Schedule - N. America
NEWS	2	MAY 01	New CAS web site launched
NEWS	3	MAY 08	CA/CAPlus Indian patent publication number format defined
NEWS	4	MAY 14	RDISCLOSURE on STN Easy enhanced with new search and display fields
NEWS	5	MAY 21	BIOSIS reloaded and enhanced with archival data
NEWS	6	MAY 21	TOXCENTER enhanced with BIOSIS reload
NEWS	7	MAY 21	CA/CAPlus enhanced with additional kind codes for German patents
NEWS	8	MAY 22	CA/CAPlus enhanced with IPC reclassification in Japanese patents
NEWS	9	JUN 27	CA/CAPlus enhanced with pre-1967 CAS Registry Numbers
NEWS	10	JUN 29	STN Viewer now available
NEWS	11	JUN 29	STN Express, Version 8.2, now available
NEWS	12	JUL 02	LEMBASE coverage updated
NEWS	13	JUL 02	LMEDLINE coverage updated
NEWS	14	JUL 02	SCISEARCH enhanced with complete author names
NEWS	15	JUL 02	CHEMCATS accession numbers revised
NEWS	16	JUL 02	CA/CAPlus enhanced with utility model patents from China
NEWS	17	JUL 16	CAPlus enhanced with French and German abstracts
NEWS	18	JUL 18	CA/CAPlus patent coverage enhanced
NEWS	19	JUL 26	USPATFULL/USPAT2 enhanced with IPC reclassification
NEWS	20	JUL 30	USGENE now available on STN
NEWS	21	AUG 06	CAS REGISTRY enhanced with new experimental property tags
NEWS	22	AUG 06	BEILSTEIN updated with new compounds
NEWS	23	AUG 06	FSTA enhanced with new thesaurus edition

NEWS EXPRESS 29 JUNE 2007: CURRENT WINDOWS VERSION IS V8.2,
CURRENT MACINTOSH VERSION IS V6.0c(ENG) AND V6.0Jc(JP),
AND CURRENT DISCOVER FILE IS DATED 05 JULY 2007.

NEWS HOURS	STN Operating Hours Plus Help Desk Availability
NEWS LOGIN	Welcome Banner and News Items
NEWS IPC8	For general information regarding STN implementation of IPC 8

Enter NEWS followed by the item number or name to see news on that specific topic.

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* * * * * STN Columbus * * * * *

FILE 'HOME' ENTERED AT 11:14:31 ON 07 AUG 2007

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=> file medline caplus biotechno biosis biotechno embase
COST IN U.S. DOLLARS                               SINCE FILE          TOTAL
                                                    ENTRY          SESSION
FULL ESTIMATED COST                               0.42              0.42
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Ca²⁺-like channels in adrenal cells.

L10 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 4
TI Intracellular Ca²⁺ store-operated influx of Ca²⁺ through TRP-R, a rat homolog of TRP, expressed in Xenopus oocytes.

L10 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 5
TI Sequence requirements of the bidirectional yeast TRP4 mRNA 3'-end formation signal.

=> d ab 1-5

L10 ANSWER 1 OF 5 MEDLINE on STN DUPLICATE 1
AB We utilized 2-aminoethoxydiphenyl borane, an agent that blocks store-operated Ca(2+) entry, as well as an antisense approach to characterize endogenous Ca(2+) entry pathways in HEK-293 cells. The thapsigargin- and carbachol-induced, but not the 1-oleoyl-2-acytyl-sn-glycerol (OAG)-induced, entry was blocked by 2-aminoethoxydiphenyl borane. Both reverse transcriptase-PCR and Western blot analyses demonstrated endogenous expression for HTRP1, HTRP3, and HTRP4 and specific suppression of mRNA levels and Trp protein levels in cells stably expressing antisense constructs. Expression of HTRP4 antisense inhibited 35% of the carbachol (CCh)-stimulated Ba(2+) entry and 46% of the OAG-stimulated Sr(2+) entry but in contrast had no effect on the thapsigargin-stimulated Ba(2+) or Sr(2+) entry. HTRP3 antisense reduced, while HTRP1 antisense had no effect on, OAG-induced Sr(2+) entry. Of greater importance, HTRP4 antisense expression, but not HTRP3 antisense expression, blocked the sustained Ca(2+) oscillations produced by low doses of CCh (15 microm), arguing that receptor-stimulated rather than store-operated channels are involved in these sustained oscillations. HTRP4 antisense also inhibited 75% of the arachidonic acid-induced Ca(2+) entry. In summary, these data suggest that HTRP4 proteins in HEK-293 cells, differing from HTRP3 and HTRP1 proteins, do not serve as functional subunits of store-operated channels but do function as subunits for CCh- and OAG-stimulated channels. Furthermore, evidence is provided for the first time for the involvement of a Trp isoform (HTRP4) in the formation of the channel responsible for both arachidonic acid-induced Ca(2+) entry and the Ca(2+) entry needed to sustain long term Ca(2+) oscillations induced by low doses of carbachol.

L10 ANSWER 2 OF 5 MEDLINE on STN DUPLICATE 2
AB Recent investigations have revealed that mammalian homologues of transient receptor potential (TRP) protein (TRP1-7) are promising candidates for Ca²⁺ entry mechanisms (or channels) associated with various metabotropic G protein-coupled receptors (GPCRs) in smooth muscle, stimulation of which generates lipid second messengers and depletes internal stores. RT-PCR and immunocytochemical experiments have demonstrated that although the level of expression varies depending on tissues, the major TRP isoforms expressed in smooth muscle are TRP4, 6 and 7. In some vascular preparations, the significant expression of TRP1 mRNA and protein is also detected. Consistent with these findings, recent functional studies using TRP6- and TRP1-specific antisense oligonucleotides and antibodies have suggested that TRP6 is the essential component of alpha1-adrenoceptor activated, store depletion-independent Ca²⁺ entry channels, while TRP1 is partly involved in Ca²⁺ entry associated with store depletion or capacitative Ca²⁺ entry. In addition, coexpression of different TRP isoforms results in the appearance of cation channels showing novel properties reminiscent of some native GPCR-activated Ca²⁺-permeable non-selective cation channels. Thus, at present, TRP proteins may be the most important clues for elucidating the molecular entities of receptor- and store-operated Ca²⁺ entry mechanisms in smooth muscle and their roles in smooth muscle functions.

- L10 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 3
 AB Mammalian TRP proteins have been implicated to function as ion channel subunits responsible for agonist-induced Ca^{2+} entry. To date, TRP proteins have been extensively studied by heterologous expression giving rise to diverse channel properties and activation mechanisms including store-operated mechanisms. However, the molecular structure and the functional properties of native TRP channels still remain elusive. Here we analyze the properties of TRP4 (CCE1) channels in their native environment and characterize TRP expression patterns and store-operated calcium currents that are endogenous to bovine adrenal cells. We show by Northern blot analysis, immunoblots, and immunohistochemistry that TRP4 transcripts and TRP4 protein are present in the adrenal cortex but absent in the medulla. Correspondingly, bovine adrenal cortex cells express TRP4 abundantly. The only other TRP transcript found at considerable levels was TRP1, whereas TRP2, TRP3, TRP5(CCE2), and TRP6 were not detectable. Depletion of calcium stores with inositol 1,4,5-trisphosphate or thapsigargin activates store-operated ion channels in adrenal cells. These channels closely resemble calcium release-activated Ca^{2+} (CRAC) channels. Expression of trp4(CCE1) cDNA in antisense orientation significantly reduces both, the endogenous CRAC-like currents and the amount of native TRP4 protein. These results demonstrate that TRP4 contributes essentially to the formation of native CRAC-like channels in adrenal cells.
- L10 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 4
 AB To elucidate whether rat transient receptor potential (TRP-R), a rat TRP4 homolog, functions as a store-operated Ca^{2+} channel (SOC), we have measured the Ca^{2+} entry after thapsigargin treatment in *Xenopus* oocytes injected with mRNA for TRP-R. While non-injected oocytes elicited an SOC response, significantly larger responses were observed in the oocytes expressing TRP-R. The oocyte-native SOC response was inhibited by injection of antisense oligodeoxyribonucleotide for mammalian TRP1. When Ca^{2+} concentration-SOC response curve was examined, the EC_{50} value was much smaller in oocytes expressing TRP-R than that of non-injected oocytes. These results suggest that TRP-R functions as SOC having higher sensitivity to external Ca^{2+} than amphibian TRP1 channel.
- L10 ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 5
 AB The yeast TRP4 3'-end formation signal functions in both orientations in an in vivo test system. We show here that the TRP4 3'-end formation element consists of two functionally different sequence regions. One region of approximately 70 nucleotides is located in the untranslated region between the translational stop codon and the major poly(A) site. The major poly(A) site is not part of this region and can be deleted without a decrease in TRP4 3'-end formation. 5' and 3' deletions and point mutations within this region affected 3'-end formation similarly in both orientations. In the center of this region the motif TAGT is located on the antisense strand. Point mutations within this motif resulted in a drastic reduce of 3'-end formation activity in both orientations. A second region consists of the 3'-end of the TRP4 open reading frame and is required for 3'-end formation in forward orientation. A single point mutation in a TAGT motif of the TRP4 open reading frame abolished TRP4 mRNA 3'-end formation in forward orientation and had no effect on the reverse orientation.

=> d 1 4

- L10 ANSWER 1 OF 5 MEDLINE on STN DUPLICATE 1
 AN 2002233850 MEDLINE
 DN PubMed ID: 11830588

TI The role of endogenous human Trp4 in regulating
 carbachol-induced calcium oscillations in HEK-293 cells.
 AU Wu Xiaoyan; Babnigg Gyorgy; Zagranichnaya Tatiana; Villereal Mitchel L
 CS Department of Neurobiology, Pharmacology, and Physiology, University of
 Chicago, Chicago, Illinois 60637, USA.
 NC GM-54500 (NIGMS)
 SO The Journal of biological chemistry, (2002 Apr 19) Vol. 277, No. 16, pp.
 13597-608. Electronic Publication: 2002-02-05.
 Journal code: 2985121R. ISSN: 0021-9258.
 CY United States
 DT Journal; Article; (JOURNAL ARTICLE)
 (RESEARCH SUPPORT, U.S. GOV'T, P.H.S.)
 LA English
 FS Priority Journals
 EM 200206
 ED Entered STN: 25 Apr 2002
 Last Updated on STN: 5 Jan 2003
 Entered Medline: 7 Jun 2002

L10 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 4
 AN 1998316914 MEDLINE
 DN PubMed ID: 9654342
 TI Intracellular Ca²⁺ store-operated influx of Ca²⁺ through TRP-R, a rat
 homolog of TRP, expressed in Xenopus oocytes.
 AU Tomita Y; Kaneko S; Funayama M; Kondo H; Satoh M; Akaike A
 CS Department of Pharmacology, Graduate School of Pharmaceutical Sciences,
 Kyoto University, Japan.
 SO Neuroscience letters, (1998 Jun 5) Vol. 248, No. 3, pp. 195-8.
 Journal code: 7600130. ISSN: 0304-3940.
 CY Ireland
 DT Journal; Article; (JOURNAL ARTICLE)
 LA English
 FS Priority Journals
 EM 199903
 ED Entered STN: 16 Mar 1999
 Last Updated on STN: 16 Mar 1999
 Entered Medline: 4 Mar 1999

=> dup rem l8
 PROCESSING COMPLETED FOR L8
 L11 22 DUP REM L8 (25 DUPLICATES REMOVED)

=> l 11 not l10
 L IS NOT A RECOGNIZED COMMAND
 The previous command name entered was not recognized by the system.
 For a list of commands available to you in the current file, enter
 "HELP COMMANDS" at an arrow prompt (=>).

=> s l11 not l10
 L12 19 L11 NOT L10

=> d 1-19 ti

L12 ANSWER 1 OF 19 MEDLINE on STN
 TI Evidence that TRPC4 supports the calcium selective I(CRAC)-like
 current in human gingival keratinocytes.
 L12 ANSWER 2 OF 19 MEDLINE on STN
 TI Pharmacological and electrophysiological characterization of
 store-operated currents and capacitative Ca(2+) entry in vascular smooth
 muscle cells.
 L12 ANSWER 3 OF 19 MEDLINE on STN

TI Functional role of TRPC proteins in native systems: implications from knockout and knock-down studies.

L12 ANSWER 4 OF 19 MEDLINE on STN

TI Molecular analysis of a store-operated and 2-acetyl-sn-glycerol-sensitive non-selective cation channel. Heteromeric assembly of TRPC1-TRPC3.

L12 ANSWER 5 OF 19 MEDLINE on STN

TI Cell-cell interaction underlies formation of fluid in the male reproductive tract of the rat.

L12 ANSWER 6 OF 19 MEDLINE on STN

TI Phospholipase cgamma1 is required for activation of store-operated channels in human keratinocytes.

L12 ANSWER 7 OF 19 MEDLINE on STN

TI Two types of store-operated Ca²⁺ channels with different activation modes and molecular origin in LNCaP human prostate cancer epithelial cells.

L12 ANSWER 8 OF 19 MEDLINE on STN

TI TRPC4 forms store-operated Ca²⁺ channels in mouse mesangial cells.

L12 ANSWER 9 OF 19 MEDLINE on STN

TI New target molecules in the drug control of blood pressure and circulation.

L12 ANSWER 10 OF 19 MEDLINE on STN

TI TRPC4 and TRPC5: receptor-operated Ca²⁺-permeable nonselective cation channels.

L12 ANSWER 11 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN

TI Inhibition of TRP channels as a treatment for cardiac hypertrophy and heart failure

L12 ANSWER 12 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN

TI Sequences of antisense oligonucleotides for modulating transient receptor potential channel 4 (TRPC4)

L12 ANSWER 13 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN

TI Modified receptors on cell membranes for the discovery of therapeutic ligands

L12 ANSWER 14 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN

TI Involvement of TRPC in the abnormal calcium influx observed in dystrophic (mdx) mouse skeletal muscle fibers

L12 ANSWER 15 OF 19 BIOSIS COPYRIGHT (c) 2007 The Thomson Corporation on STN

TI Molecular identification of store-operated calcium channels (SOC) in mouse glomerular mesangial cells (MMC).

L12 ANSWER 16 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

TI Receptor-operated cation channels formed by TRPC4 and TRPC5.

L12 ANSWER 17 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

TI Newly emerging Ca(2+) entry channel molecules that regulate the vascular tone.

L12 ANSWER 18 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

TI TRPC4 forms store-operated Ca(2+) channels in mouse mesangial

cells.

L12 ANSWER 19 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

TI Molecular linkage of TRP proteins to smooth muscle receptor-operated Ca(2+)-permeable cationic channels.

=> d ab 10 11 12 9 16

L12 ANSWER 10 OF 19 MEDLINE on STN

AB The seven mammalian channels from the classical (TRPC) subfamily of transient receptor potential (TRP) channels are thought to be receptor-operated cation channels activated in a phospholipase C (PLC)-dependent manner. Based on sequence similarity, TRPC channels can be divided into four subgroups. Group 4 comprises TRPC4 and TRPC5, and is most closely related to group 1 (TRPC1). The functional properties observed following heterologous expression of TRPC4 or TRPC5 in mammalian cells are contradictory and, therefore, controversial. In our hands, and in several independent studies, both channels, probably as homotetramers, form receptor-operated, Ca2+-permeable, nonselective cation channels activated independently of inositol 1,4,5-trisphosphate (InsP(3)) receptor activation or Ca2+ store-depletion. As heteromultimers with TRPC1, TRPC4 and TRPC5 form receptor-operated, Ca2+-permeable, nonselective cation channels with biophysical properties distinct from homomeric TRPC4 or TRPC5. In other studies, TRPC4 and TRPC5 have been shown to be store-operated channels, with moderate to high Ca2+ permeabilities. At present there is no clear explanation for these major differences in functional properties. To date, little is known as to which native cation channels are formed by TRPC4 and TRPC5. Endothelial cells from TRPC4(-/-) mice lack a highly Ca2+-permeable, store-dependent current, and data support a role for TRPC4 in endothelium-mediated vasorelaxation. A similar current in adrenal cortical cells is reduced by TRPC4 antisense. From similarities in the properties of the currents and expression of appropriate isoforms in the tissues, it is likely that heteromultimers of TRPC1 and TRPC4 or TRPC5 form receptor-operated nonselective cation channels in central neurones, and that TRPC4 contributes to nonselective cation channels in intestinal smooth muscle.

L12 ANSWER 11 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN

AB The invention provides methods for treating and preventing cardiac hypertrophy and heart failure. MEF-2, NF-AT3, calcineurin, MCIP, and Class II HDACs have been shown to have a major role in cardiac hypertrophy and heart disease, and inhibition of many of these factors or the pathways mediated by these factors has been shown to have a beneficial, anti-hypertrophic effect. The invention provides a link between these factors and the pathways they mediate through a family of non-voltage gated channels called TRP channels. The invention further demonstrates that inhibitors of TRP channels can inhibit or treat heart failure and cardiac hypertrophy.

L12 ANSWER 12 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN

AB The invention provides sequences of antisense oligonucleotides, compns. and method useful for modulating the expression of TRPC4. The compns. comprise antisense oligonucleotides targeted to nucleic acids encoding TRPC4. Antisense oligonucleotides targeted to TRPC4 mRNA could be used therapeutically to reduce the level of TRPC4 receptors in a patient suffering from chronic pain.

L12 ANSWER 9 OF 19 MEDLINE on STN

AB Ion channels play a pivotal role in blood pressure regulation. Amongst

them, much attention has been directed to dihydropyridine (DHP)-sensitive (L-type) voltage-dependent Ca(2+) channels (VDCCs) and iberiotoxin-sensitive Ca(2+)-dependent K(+) channels which are distributed over the whole vascular tree and contribute to vascular tone regulation. Recent advances in vascular electrophysiology have, however, added novel and interesting molecules to this repertoire. In small mesenteric arterioles, the predominant VDCC phenotype is not L-type but DHP-insensitive, high voltage-activated VDCCs that exhibit unique properties distinguishable from those of hitherto-known VDCCs. Surprisingly, mibefradil, a well-known T-type selective blocker potentially inhibits these channels, and the use of this blocker has indicated that Ca(2+) entry through these channels may be one of the important determinants of peripheral vascular tone. Another new candidate likely involved in blood pressure control is the mammalian homologue of Drosophila transient receptor potential (TRP) protein, including TRPC4 and TRPC6. Experiments in genetically engineered TRPC4-deficient mice have suggested that expression of TRPC4 is indispensable for agonist-induced Ca(2+) entry in endothelial cells and production of nitric oxide and vasorelaxation. TRPC6 is likely to contribute to sustained Ca(2+) entry into vascular smooth muscle cells activated by stimulation of sympathetic nerves and elevation of intravascular pressure. Antisense oligonucleotide experiments have suggested that this protein is an essential component of alpha1-adrenoceptor activated and mechanosensitive cation channels in some vascular tissues. This review overviews what is known about the role of ionic channels in blood pressure control with main focus on the above-mentioned new molecules as promising targets for drug discovery and development.

L12 ANSWER 16 OF 19 EMBASE COPYRIGHT (c) 2007 Elsevier B.V. All rights reserved on STN

AB TRPC4 and TRPC5 form cation channels that contribute to phospholipase C-dependent Ca(2+) entry following stimulation of G-protein-coupled receptors or receptor tyrosine kinases. Surprisingly, in different studies, TRPC4 and TRPC5 have been shown to form either store-operated channels with a relatively high Ca(2+) permeability, or nonselective cation channels activated independently of store depletion. In this review, we summarize and discuss data on the regulation and permeability properties of TRPC4 and TRPC5, and data on native channels that might be composed of these isoforms. .COPYRGHT. Springer-Verlag 2005.

=> d 9 10 12

L12 ANSWER 9 OF 19 MEDLINE on STN

AN 2003247345 MEDLINE

DN PubMed ID: 12769645

TI New target molecules in the drug control of blood pressure and circulation.

AU Inoue Ryuji; Mori Yasuo

CS Department of Pharmacology, Graduate School of Medical Sciences, Kyushu University, Fukuoka 812-8582, Japan.. inouery@pharmaco.med.kyushu-u.ac.jp

SO Current drug targets. Cardiovascular & haematological disorders, (2003 Mar) Vol. 3, No. 1, pp. 59-72. Ref: 115

Journal code: 101123341. ISSN: 1568-0061.

CY Netherlands

DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)

LA English

FS Priority Journals

EM 200306

ED Entered STN: 29 May 2003

Last Updated on STN: 25 Jun 2003

Entered Medline: 24 Jun 2003

L12 ANSWER 10 OF 19 MEDLINE on STN
AN 2003243443 MEDLINE
DN PubMed ID: 12765689
TI TRPC4 and TRPC5: receptor-operated Ca²⁺-permeable nonselective
cation channels.
AU Plant Tim D; Schaefer Michael
CS Institut fur Pharmakologie, Freie Universitat Berlin, Thielallee 67-73,
14195 Berlin, Germany.. tplant@zedat.fu-berlin.de
SO Cell calcium, (2003 May-Jun) Vol. 33, No. 5-6, pp. 441-50. Ref: 54
Journal code: 8006226. ISSN: 0143-4160.
CY Scotland: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
(RESEARCH SUPPORT, NON-U.S. GOV'T)
General Review; (REVIEW)
LA English
FS Priority Journals
EM 200402
ED Entered STN: 28 May 2003
Last Updated on STN: 25 Feb 2004
Entered Medline: 24 Feb 2004

L12 ANSWER 12 OF 19 CAPLUS COPYRIGHT 2007 ACS on STN
AN 2003:551623 CAPLUS
DN 139:95492
TI Sequences of antisense oligonucleotides for modulating transient
receptor potential channel 4 (TRPC4)
IN Shuster, Samuel J.; Arvidsson, Ulf N. G.; Stone, Laura S.; Zhang,
Hong-yan; Hart, Lucy Vulchanova
PA Algos Therapeutics, Inc., USA
SO PCT Int. Appl., 37 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
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PI	WO 2003057843	A2	20030717	WO 2002-US41751	20021231
	WO 2003057843	A3	20031218		
	WO 2003057843	A8	20040506		
	W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NO, NZ, OM, PH, PL, PT, RO, RU, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZM, ZW				
	RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
	AU 2002364610	A1	20030724	AU 2002-364610	20021231
	US 2006194750	A1	20060831	US 2004-500493	20041203
PRAI	US 2001-346171P	P	20011231		
	WO 2002-US41751	W	20021231		

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SINCE FILE

TOTAL

ENTRY

SESSION

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